optimized for mammary epithelial cell growth or embedded in the basement membrane extract, Matrigel. Immunocytochemistry and laser scanning confocal microscopy was performed on the cell populations to detect differentiation markers including the water channel protein, aquaporin 5, which is a proacinar/acinar marker. Epithelial organoids derived from cell clusters did not maintain expression of aquaporin 5 after seven days in culture with simple media. In the presence of Matrigel or the mammary growth media, the epithelial phenotype was preserved but the proacinar marker was lost. However, in co-cultures of epithelium with primary mesenchyme but not with NIH 3T3 cells, proacinar differentiation was detected as aquaporin 5 protein expression. These results indicate that specific heterotypic cell-cell interactions are required to maintain mouse SMG proacinar cell differentiation. Mesenchymal factors will be identified that are permissive to promote acinar differentiation in future work. The mesenchymal factors that promote acinar differentiation could be incorporated into scaffolds for application in therapeutics that restore gland function.

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Board Number: B1426

The balance between N-cadherin and E-cadherin orchestrates major neuroectodermal cell fate choices.

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Numerous cadherin proteins, including N-cadherin (Ncad), E-cadherin (Ecad), Cadherin-11 (Cad11) and Cadherin-7 (Cad7), are expressed in the developing neural plate as well as in neural crest cells as they delaminate from the newly closed neural tube. To clarify whether these proteins function independently or coordinately during development, we examined their relative expression in the cranial region of chick embryos. The results revealed surprising overlap of Ecad, Ncad and Cad7 in the neural tube, suggesting possible heterotypic interactions. Using a proximity ligation assay and co-immunoprecipitation to test this hypothesis, we found that Ncad formed heterophilic complexes in the developing neural tube with Ecad. We also determined that modulation of either Ncad or Ecad levels led to reciprocal gain or reduction of the other cadherin protein. Altering levels of the two cadherin proteins affected the early fate specification of ectodermal derivatives, forcing an aberrant choice between neural crest and epidermal cells. Finally, we identified that the availability of β-catenin plays a critical role in maintaining the balance between Ncad and Ecad in early development since co-expression of activated β-catenin rescues the Ncad-overexpression phenotype. These results suggest that β-catenin-mediated balance of Ncad and Ecad proteins is critical for the normal development of the three ectodermal derivatives.

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RhoGTPases and Mesenchymal-to-Epithelial Transitions: same legs (actin), but different shoes (cadherins vs. integrins).

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The ability of cells to transition between a migratory mesenchymal state and an epithelial cell-cell adherent state and vice versa is a hallmark of metazoan biology and is necessary for a number of developmental processes including tumor metastasis. The mechanisms that regulate such transitions remain unclear, but require coordination of the cell-substrate adhesion machinery (integrin pathways),